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AMSTER, ROTHSTEIN & EBENSTEIN LLP
90 PARK AVENUE
NEW YORK, NY 10016

EXAMINER	
DUNSTON, JENNIFER ANN	
ART UNIT	PAPER NUMBER
1636	
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Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary	Application No.	Applicant(s)	
	10/586,892	LAWRENCE ET AL.	
	Examiner	Art Unit	
	Jennifer Dunston	1636	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *19 March 2010*.

2a)☒ This action is **FINAL**. 2b)☐ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1-16,18-31 and 35-38* is/are pending in the application.

4a) Of the above claim(s) *5,7,8,22,23,26,31 and 35-38* is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1-4,6,9-16,18-20 and 27-30* is/are rejected.

7)☒ Claim(s) *21,24 and 25* is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☒ The drawing(s) filed on *20 July 2006* is/are: a)☒ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date *3/19/2010*.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED ACTION

This action is in response to the amendment, filed 3/19/2010, in which claims 17 and 32-34 were canceled, and claims 1, 7, 9, 22, 24, 37 and 38 were amended. Claims 1-16, 18-31 and 35-38 are pending.

Applicant's arguments have been thoroughly reviewed, but are not persuasive for the reasons that follow. Any rejections and objections not reiterated in this action have been withdrawn. **This action is FINAL.**

Election/Restrictions

Applicant's election without traverse of gentamicin as the species of agent effective to suppress a mutation/correct a defect caused by a mutation, and thioguanine as the species of agent effective to increase transcription of a gene in the reply filed on 4/13/2009 is acknowledged.

In the amendment filed 3/19/2010, claim 31 was amended to include the limitations of withdrawn claim 33. Further, the claim does not read on the administration of gentamicin as the agent effective to suppress a mutation/correct a defect caused by a mutation. Thus, claim 31 and claims that depend therefrom read on a withdrawn species.

Claims 5, 7-8, 22-23, 26, 31 and 35-38 are withdrawn from further consideration pursuant to 37 CFR 1.142(b) as being drawn to a nonelected species, there being no allowable generic or linking claim. Election was made **without** traverse in the reply filed on 4/13/2009.

Claims 1-4, 6, 9-16, 18-21, 24, 25 and 27-30 are under consideration.

Information Disclosure Statement

Receipt of an information disclosure statement, filed on 3/19/2010, is acknowledged. The signed and initialed PTO 1449 has been mailed with this action.

Claim Rejections - 35 USC § 112

The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

Claims 1-4, 6, 9-16, 18-20 and 27-30 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for treating ataxia telangiectasia in a subject, comprising administering to the subject an amount of gentamicin effective to suppress a premature stop codon in the *atm* gene, and an amount of a fluorinated quinolone or thioguanine effective to increase transcription of the *atm* gene, does not reasonably provide enablement for making and using an agent effective to increase transcription of any gene disrupted by the presence of a premature stop codon, and making and using the method to enhance production of a protein disrupted by any genetic mutation where a compound is administered to suppress the genetic mutation and/or correct a defect caused by the mutation, and a compound is administered to increase transcription of the gene. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the invention commensurate in scope with these claims. This rejection was made in the Office action mailed 12/28/2009 and has been rewritten to address the amendments to the claims in the reply filed 3/19/2010.

Enablement is considered in view of the Wands factors (MPEP 2164.01(A)). These include: nature of the invention, breadth of the claims, guidance of the specification, the existence of working examples, state of the art, predictability of the art and the amount of experimentation necessary. All of the Wands factors have been considered with regard to the instant claims, with the most relevant factors discussed below.

Nature of the invention: Claims 1-4, 6, 9-16, 18-20 and 27-30 are drawn to a method for enhancing production in a subject of a functional protein from a gene disrupted by the presence of a premature stop codon in the coding region of the gene, comprising administering to the subject an amount of an agent effective to suppress the premature stop codon and an amount of an agent effective to increase the transcription of the gene, wherein the agent that is effective to increase transcription of the gene is a fluorinated quinolone or thioguanine, and wherein the agent that suppresses the premature stop codon is administered at a dose lower than the dose that would be required to produce the same amount of functional protein in the absence of the agent that increases transcription. Dependent claims limit the agent effective to suppress the premature stop codon to an aminoglycoside antibiotic (claim 2) or gentamicin (elected species, claims 3 and 4). The dependent claims limit the agent that increases transcription of the gene to an agent that activates a promoter of the gene (claim 6), or more specifically thioguanine (claim 9). Dependent claims 10-16 are directed to the level of enhancement obtained by the agents. Claim 18 requires the lower dose of claim 1 to result in decreased toxicity. Claims 19-20 require the disruption of the gene to be associated with a genetic disorder (claim 19), or a genetic disorder selected from the group consisting of thalassemia, hemophilia A, hemophilia B, von Willebrand's disease, a p53 related cancer or disorder, a colorectal cancer, cystinosis, cystic fibrosis,

Duchenne muscular dystrophy, Tay-Sachs disease, Wilms tumor, retinoblastoma, neurofibromatosis, ataxia telangiectasia, Hurler's syndrome, mucopolysaccharidosis I, and late infantile neuronal ceroid lipofuscinosis (claim 20). Claim 27 requires the genetic disorder to be treated. Claim 28 requires the disrupted gene to be a tumor suppressor gene, and claim 29 limits the tumor suppressor gene to BRCA1, BRCA2, PTEN, NF1, NF2, MLH1, MLH2, VHL, WT1, TSC1, TSC2 and/or ATM. Claim 30 requires enhanced production of the functional protein to be effective in treating a tumor in a subject.

The nature of the invention is complex in that thioguanine (elected species) must activate expression of a gene disrupted by a premature stop codon, including any gene that is associated with a genetic disorder, any gene associated with the specific genetic disorders recited in the claims, any tumor suppressor gene, and any tumor suppressor gene recited in the claims. Thioguanine must also be capable of acting, directly or indirectly, on the promoter of the gene disrupted by a premature stop codon.

Breadth of the claims: The claims broadly encompass the treatment of any genetic disorder. The claims require thioguanine to be capable of increasing the transcription of any gene that may contain a premature stop codon. The complex nature of the subject matter of this invention is greatly exacerbated by the breadth of the claims.

Guidance of the specification and existence of working examples: The specification teaches that the termination of protein synthesis is signaled by the nucleic acid stop (nonsense) codons UAA, UAG, and UGA, and nonsense mutations occur when a sense codon is changes into one of the three stop codons (e.g., paragraph [0005]). Nonsense mutations result in the premature termination of protein synthesis, and the truncation or absence of a key protein

product and are associated with a host of genetic diseases, including thalassemia (α-globin and β-globin genes), hemophilia A and B (factor VIII and factor IX genes), von Willebrand's disease (vWF gene), p53 related cancers (p53 gene), colorectal cancers *(APC,* MSH1, and MSHZ genes), cystic fibrosis (CFTR gene), Duchenne muscular dystrophy (dystrophin gene), Tay-Sachs disease (hexosaminidase A gene), Wilms tumor (Wtl gene), retinoblastoma (Rb gene), neurofibromatosis (IW1 and NFZ genes), ataxia telangiectasia *(atm* gene), the lysosomal storage disease mucopolysaccharidosis I (IDUA gene), Hurler's syndrome, cystinosis, and late infantile neuronal ceroid lipofuscinosis. Alternatively, the nonsense mutation can occur in a tumor suppressor gene, such as BRCA1, BRCA2, PTEN, NF1, NF2, MLH1, MLH2, VHL, WTI, TSCl, TSC2, and ATM (e.g., paragraph [0023]).

The specification teaches that it was known in the art that gentamicin and other aminoglycoside antibiotics can suppress premature stop codon arrest by inducing the ribosome to read past the nonsense mutation via insertion of a random amino acid by a noncognate tRNA (e.g., paragraph [0006]). The specification acknowledges that the prior art teaches the use of aminoglycosides to suppress nonsense mutations in human cell lines and animal models of Hurler's syndrome, Duchenne muscular dystrophy, late infantile neuronal ceroid lipofuscinosis, cystinosis, cystic fibrosis, mucopolysaccharidosis I, and P863 gene related disorders (e.g., paragraph [0006]). Moreover, gentamicin has also been used in patients with cystic fibrosis and Duchenne muscular dystrophy (e.g., paragraphs [0006] and [0013]).

With regard to the compound used to suppress a premature stop codon, the specification teaches the use of an aminoglycoside antibiotic, or PTC124 (e.g., paragraph [0012]).

Aminoglycoside antibiotics include gentamicin, geneticin, paromomycin, hygromycin, G-418, kanamycin, anlikacin and tobrarnycin (e.g., paragraph [0012]).

The specification envisions using an agent that increases transcription of a gene, such as an agent that activates a promoter of a gene (e.g., paragraph [0014]). The specification envisions the use of agents that directly or indirectly activate a promoter (e.g., paragraph [0014]). The specification states that activators can be identified by one skilled in the art using methods similar to those disclosed in the specification (e.g., paragraph [0015]). For example, the specification envisions the identification of activators by (i) cloning the promoter of the gene; (ii) attaching the promoter to a luciferase cDNA; (iii) transfecting the promoter/luciferase construct into an appropriate cell line; and (iv) using the cell line to simultaneously screen multiple candidate activators of the promoter (e.g., paragraph [0015]). The specification envisions screening a library of nearly 400 drugs approved by the Federal Drug Administration (FDA) (e.g., paragraph [0015]).

The working examples teach the application of the abovementioned screening assay for agents that increase transcription of a gene to the *atm* gene, which is mutated in ataxia telangiectasia. The specification notes that high glucose levels up-regulate ATM message and protein levels (e.g., paragraph [0037]). With regard to the compounds identified by the assay, the specification states, "several FDA approved drugs activate the *atm* promoter, including the fluorinated quinolone ofloxacin" (paragraph [0037]). The specification teaches that fluorinated quinolones are known to target eukaryotic topoisomerase II, an enzyme that catalyzes the interpenetration of DNA strands by introducing transient double strand breaks, and the possible mechanism by which ofloxacin activates the *atm* promoter is via the induction of double strand

break recognition or repair (e.g., paragraph [0037]). The specification notes that if this mechanism is correct, then the production of ATM protein would need to be sufficient to offset any drug-induced double strand breaks (e.g., paragraph [0037]). The specification also discloses that the antimetabolite thioguanine also increases *atm* promoter activity (e.g., paragraph [0037]). The specification alludes to other compounds that induce *atm* promoter activity but does not disclose the compounds by name or structure. The effects of ofloxacin; geneticin; gentamicin; ofloxacin and gentamicin; thioguanine and gentamicin; and ofloxacin and geneticin were studied in a cell culture model. Each of the compound(s) tested increased the expression of functional protein from the coding sequence linked to an *atm* promoter. The effects of the compounds appear to be specific to the *atm* promoter. The specification does not provide evidence that the actions of ofloxacin and thioguanine can be extrapolated to the promoter of any gene that contains a premature stop codon.

Predictability and state of the art: It would have been unpredictable to use thioguanine to increase the transcription of any gene containing a premature stop codon. The specification teaches the screening of compounds to identify compounds that increase *atm* promoter activity. The screen disclosed in the present specification identified ofloxacin, a fluorinated quinolone that introduces double strand breaks, as capable of increasing *atm* promoter activity (e.g., paragraph [0037]). The specification also notes that thioguanine is capable of increasing *atm* promoter activity (e.g., paragraph [0037]). The specification cites the Bokkerink et al reference (Bokkerink et al. Biochemical Pharmacology, Vol. 45, No. 7, pages 1455-1463, 1993), which teaches that thioguanine introduces chromosomal breaks (e.g., paragraph bridging pages 1460-1461). However, there is no evidence that chromosomal breaks induce the expression of each of

the following genes: genes that may contain a premature stop codon; genes associated with thalassemia (α-globin and β-globin genes), hemophilia A and B (factor VIII and factor IX genes), von Willebrand's disease (vWF gene), p53 related cancers (p53 gene), colorectal cancers *(APC,* MSH1, and MSHZ genes), cystic fibrosis (CFTR gene), Duchenne muscular dystrophy (dystrophin gene), Tay-Sachs disease (hexosaminidase A gene), Wilms tumor (Wtl gene), retinoblastoma (Rb gene), neurofibromatosis (IW1 and NFZ genes), the lysosomal storage disease mucopolysaccharidosis I (IDUA gene), Hurler's syndrome, cystinosis, and late infantile neuronal ceroid lipofuscinosis; or tumor suppressor genes, such as BRCA1, BRCA2, PTEN, NF1, NF2, MLH1, MLH2, VHL, WTI, TSCl and TSC2. The teachings of Bokkerink et al support the unpredictability of the claimed invention. Bokkerink et al teach that the most important mechanism of cytotoxicity of 6-Mercaptopurine (6MP) was shown to be incorporation as 6-thioguanine (6TG) deoxyribonucleotides into DNA; however, metabolism of 6MP in cells also results in decreased RNA synthesis due to inhibition of the first enzyme in purine *de novo* synthesis, which results in decreased availability of ribonucleotides (e.g., page 1455, right column; page 1458, left column, full paragraph; page 1459, right column, last full paragraph; Figure 6). The prior art also teaches that 6-thioguanine is a ribonucleotide derivative that inhibits the first enzyme of purine biosynthesis (McCollister et al. The Journal of Biological Chemistry, Vol. 239, No. 5, pages 1560-1563, May 1964; e.g., page 1563, left column, Summary). Except for the ATM gene, one would expect the administration of thioguanine to generally decrease the transcription of any gene. Accordingly, it would have been unpredictable to use thioguanine to increase the transcription of any gene.

Amount of experimentation necessary: The claims require the administration of thioguanine to be capable of increasing the transcription of any gene comprising a premature stop codon and to activate the promoter of any such gene. Except for the *atm* gene, the gene promoters capable of being activated by thioguanine have not been defined in the specification or prior art. One would have been required to assay thioguanine for the ability to increase the expression of genes mutated in thalassemia (α-globin and β-globin genes), hemophilia A and B (factor VIII and factor IX genes), von Willebrand's disease (vWF gene), p53 related cancers (p53 gene), colorectal cancers *(APC,* MSH1, and MSHZ genes), cystic fibrosis (CFTR gene), Duchenne muscular dystrophy (dystrophin gene), Tay-Sachs disease (hexosaminidase A gene), Wilms tumor (Wtl gene), retinoblastoma (Rb gene), neurofibromatosis (IW1 and NFZ genes), the lysosomal storage disease mucopolysaccharidosis I (IDUA gene), Hurler's syndrome, cystinosis, and late infantile neuronal ceroid lipofuscinosis, or any tumor suppressor gene, such as BRCA1, BRCA2, PTEN, NF1, NF2, MLH1, MLH2, VHL, WTI, TSCl and TSC2. Because each gene has a distinct promoter and there is no disclosure of a common thioguanine responsive element in each gene, and the prior art teaches a general reduction in transcription upon administration of thioguanine, it would require a large amount of experimentation to identify the operable embodiments that fall within the scope of what is claimed.

In view of the breadth of the claims and the lack of guidance provided by the specification as well as the unpredictability of the art, the skilled artisan would have required an undue amount of experimentation to make and/or use the claimed invention. Therefore, claims 1-4, 6, 9-16, 18-20 and 27-30 are not considered to be fully enabled by the instant specification.

Response to Arguments - 35 USC § 112

The rejection of claims 31, 35-36 and 38 under 35 U.S.C. 112, second paragraph, is moot in view of the amendment, filed 3/19/2010, to amend the claims such that the do not read on the elected species.

The rejection of claims 31, 35-36 and 38 under 35 U.S.C. 112, first paragraph (scope of enablement), is moot in view of the amendment, filed 3/19/2010, to amend the claims such that the do not read on the elected species.

The rejection of claim 17 under 35 U.S.C. 112, first paragraph (scope of enablement) is moot in view of Applicant's cancellation of the claim in the reply filed 3/19/2010.

The rejection of claims 21, 24 and 25 under 112, first paragraph (scope of enablement), has been withdrawn in view of Applicant's amendment to the claims in the reply filed 3/19/2010. The claims are commensurate in scope with the subject matter indicated as enabled on page 4 of the Office action mailed 12/28/2009.

With respect to the rejection of claims 1-4, 6, 9-16, 18-20 and 27-30 under 35 U.S.C. 112, first paragraph, Applicant's arguments filed 3/19/2010 have been fully considered but they are not persuasive.

At page 13 paragraph 3, the response notes that the claims have been amended to require that the agent that is effective to increase transcription of the gene is a fluorinated quinolone or thioguanine. At page 13, paragraph 4, the response notes that many agents are known to suppress premature stop codons. At page 13, paragraph 5, the response notes that many diseases were know to be associated with nonsense mutations. The paragraph bridging pages 13-14 is directed to withdrawn claim 31.

These arguments are not persuasive. The rejection of record was not based upon the lack of known agents to suppress premature stop codons, or the lack of diseases known to be associated with nonsense mutations. Rather, the claims encompassed the use of a compound defined primarily by function, which was a compound capable of increasing transcription of any gene that contains a premature stop codon, or more specifically any gene mutated in thalassemia (α-globin and β-globin genes), hemophilia A and B (factor VIII and factor IX genes), von Willebrand's disease (vWF gene), p53 related cancers (p53 gene), colorectal cancers *(APC,* MSH1, and MSHZ genes), cystic fibrosis (CFTR gene), Duchenne muscular dystrophy (dystrophin gene), Tay-Sachs disease (hexosaminidase A gene), Wilms tumor (Wtl gene), retinoblastoma (Rb gene), neurofibromatosis (IW1 and NFZ genes), the lysosomal storage disease mucopolysaccharidosis I (IDUA gene), Hurler's syndrome, cystinosis, and late infantile neuronal ceroid lipofuscinosis, or any tumor suppressor gene, such as BRCA1, BRCA2, PTEN, NF1, NF2, MLH1, MLH2, VHL, WTI, TSCl and TSC2. In the amendment filed 3/19/2010 the claims were amended to require thioguanine (elected species) to be capable of increasing the transcription of each of these genes. The specification provides evidence that thioguanine is capable of increasing transcription of the *atm* gene, which is mutated in ataxia telangiectasia. However, there is no evidence that thioguanine is capable of increasing the transcription of any of the other claimed genes. The specification discloses that the ability of a compound to regulate a given promoter must be empirically determined, and there is no evidence that thioguanine regulates any promoter other than the *atm* promoter. Furthermore, the teachings of the prior art support a role for thioguanine in generally decreasing RNA synthesis. Accordingly, one would

not have been able to use thioguanine to increase the transcription of any gene comprising a premature stop codon, as required by the claims.

For these reasons, and the reasons made of record in the previous office actions, the rejection is maintained.

The rejection of claims 31, 35-36 and 38 under 35 U.S.C. 112, first paragraph (written description), is moot in view of the amendment, filed 3/19/2010, to amend the claims such that the do not read on the elected species.

The rejection of claim 17 under 35 U.S.C. 112, first paragraph (scope of enablement) is moot in view of Applicant's cancellation of the claim in the reply filed 3/19/2010.

The rejection of claims 1-4, 6, 10-16, 18-21, 25, 27-30 under 35 U.S.C. 112, first paragraph (written description), has been withdrawn in view of Applicant's amendment to the claims in the reply filed 3/19/2010. The claims require the agent effective to increase transcription of the gene to be thioguanine (elected species). Thus, the structure of the agent is described.

Response to Arguments - 35 USC § 102

The rejection of claims 1, 6, 10-16, 19-21, 27-31, 35-36 and 38 under 35 U.S.C. 102(e) as being anticipated by Wilde et al has been withdrawn in view of Applicant's amendment to the claims in the reply filed 3/19/2010.

Response to Arguments - 35 USC § 103

The rejection of claims 2-4 and 25 under 35 U.S.C. 103(a) as being unpatentable over Wilde et al has been withdrawn in view of Applicant's amendment to the claims in the reply filed 3/19/2010.

Conclusion

No claims are allowed.

Claims 21, 24 and 25 are objected to as being dependent upon a rejected base claim, but would be allowable if rewritten in independent form including all of the limitations of the base claim and any intervening claims.

Applicant's amendment necessitated the new ground(s) of rejection presented in this Office action. Accordingly, **THIS ACTION IS MADE FINAL**. See MPEP § 706.07(a). Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the date of this final action.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Jennifer Dunston whose telephone number is (571)272-2916. The examiner can normally be reached on M-F, 9 am to 5 pm.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Christopher Low can be reached on 571-272-0951. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

/Jennifer Dunston/
Primary Examiner
Art Unit 1636